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Oyster Mortalities, with Particular Reference to Chesapeake Bay and the Atlantic Coast of North America

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CARL J. SINDERMANN

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CONTENTS

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By

CARL J. SINDERMANN, Fishery Biologist

Bureau of Commercial Fisheries Biological Laboratory
Oxford, Maryland 21654

ABSTRACT

A number of recent mass mortalities of oysters of the Middle Atlantic States and elsewhere in the world have been attributed to the effects of disease. Oyster production in Delaware Bay and lower Cheaspeake Bay has been seriously reduced during the past decade by an epizootic of a protozoan pathogen, Minchinia nelsoni. Other recent disease-associated mortalities of oysters have occurred in the Gulf of Saint Lawrence and the Gulf of Mexico. Man may have aided spread of diseases by transfers and overcrowding of beds. Reduction of this threat to oyster production could be effected by quarantines, development of disease-resistant strains of oysters, and use of environmental barriers (such as low salinity) to the pathogens involved.

INTRODUCTION

Many of the great fisheries of the world are characterized by fluctuations in supply. The causes of such fluctuations have been much discussed but rarely determined. Accused as causes of reduction in abundance of commercial marine species have been overfishing, failure of spawning, sudden and drastic changes in the environment, and a host of other factors. Disease has received limited attention as a critical factor in population control. The fact that marine animals get sick and die, often in vast numbers, has been accepted but then often ignored. Events have occurred in the oyster industry in recent decades, however, that force us to examine disease as a cause of mass mortalities of epic proportions. This paper summarizes recent information about mass mortalities and their effects on the American oyster (Crassostrea virginica) industry.

[1] Based on material presented at the 22d meeting of the American Fisheries Advisory Committee, Irvington, Va., October 24, 1966.

OYSTER MORTALITIES IN CHESAPEAKE BAY

The history and present status of the Chesapeake Bay oyster fishery can provide important background information on a declining resource. For most of this century, about 50 percent of the total national harvest of oysters has come from the complex estuarine system that we call Chesapeake Bay. The somewhat dismal picture of national oyster production is shown in figure 1. Landings descended erratically from a peak of 170 million pounds of meats in the late 1890's to the present level of about 60 million pounds--which includes about 10 million pounds of Pacific oysters, Crassostrea gigas (Engle, 1966). Of this total, Chesapeake Bay production once exceeded 100 million pounds but is now only about 20 million pounds. The reasons for this downward trend for the past 70 years are complex. Some reasons given are hotly debated, but they certainly include such factors as loss of growing areas because of pollution, intensive fishing on a natural stock without adequate management, and the lack of development of anything but the most rudimentary cultivation.

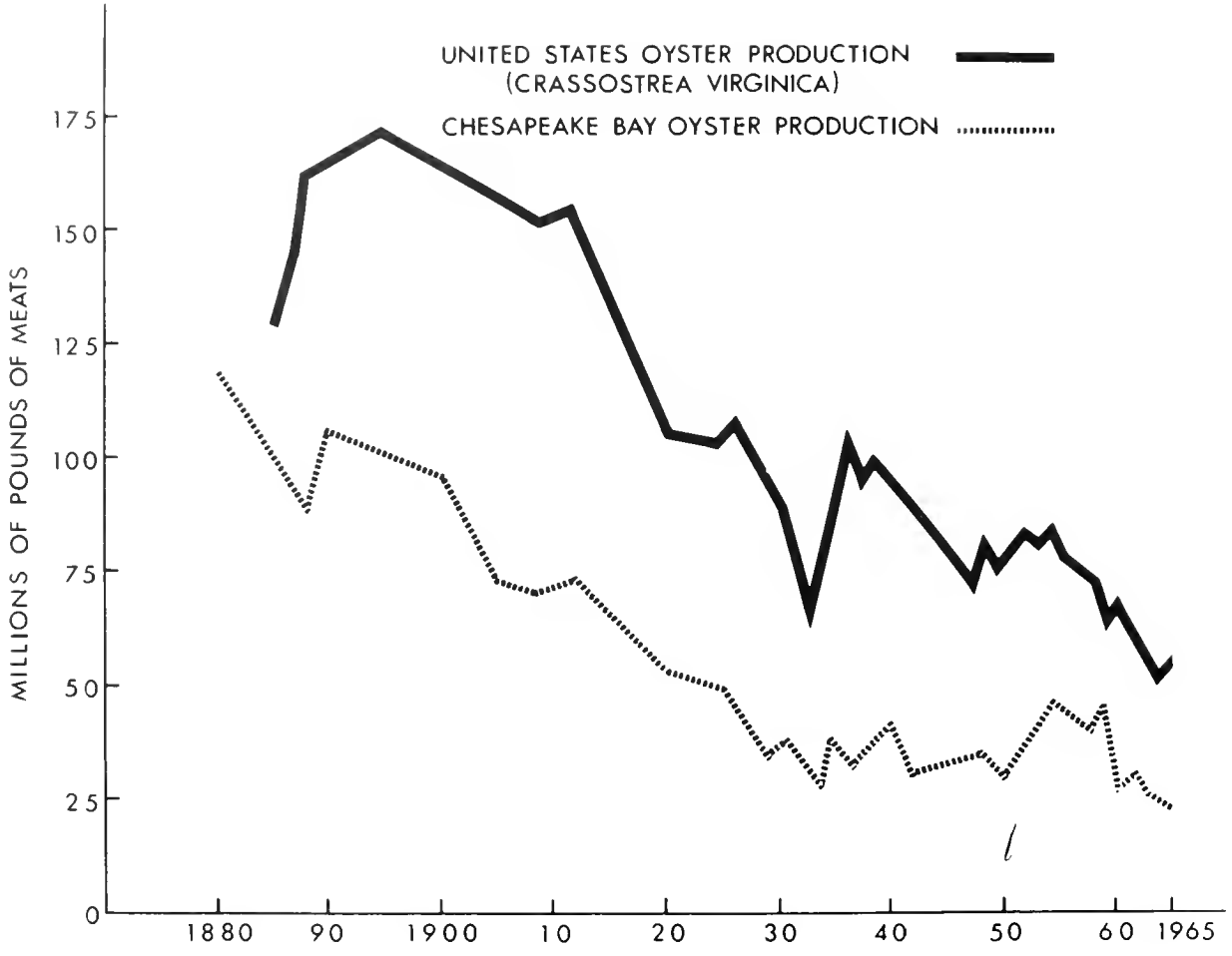

Figure 1.--Production of oysters (Crassostrea virginica) in Chesapeake Bay and in the United States, 1880 to 1965. Modified from Galtsoff (1956) and Engle (1966).

The oyster fisheries in Maryland and Virginia waters of the Bay are very different (fig 2). Most of Maryland production is from public beds, and the State has carried on a vigorous large-scale program of shell planting and seed transfer since 1961. Virginia production, on the other hand, is principally from private beds, most of which have depended on the James River as a seed source.

Total oyster production in Chesapeake Bay over the last two decades (fig 3) shows the same distressing downward trend from 1954 to 1964, despite Maryland's oyster shell planting program and Virginia's private planting. Production has slipped from about 40 million pounds to only 20 million during this period. We have good evidence that disease has been responsible for much of the decline since 1960, particularly in Virginia waters.

The disease outbreak responsible for oyster mortalities in Chesapeake Bay actually began several years earlier--probably about 1955--in Delaware Bay. The oyster industry there was almost completely destroyed by 1959 and has shown little recovery (fig 4). Production in New Jersey waters of Delaware Bay dropped from a high of 7.5 million pounds of shucked meats in 1953 to a low of only one-third of a million pounds in 1960. Mortalities in Delaware Bay reached 85 percent in certain beds.

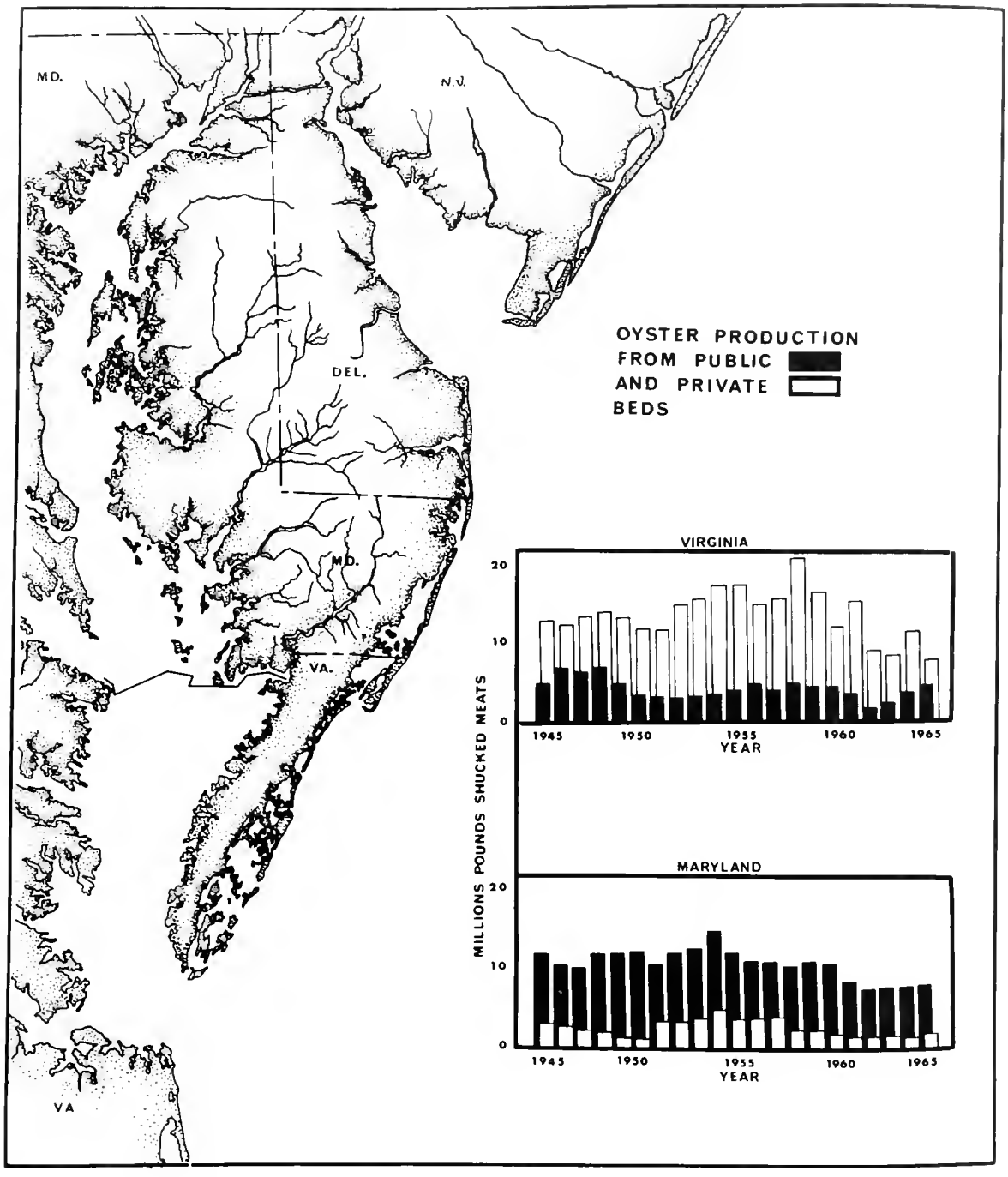

Figure 2.--Oyster production from public and private beds, Maryland and Virginia, 1945-65.

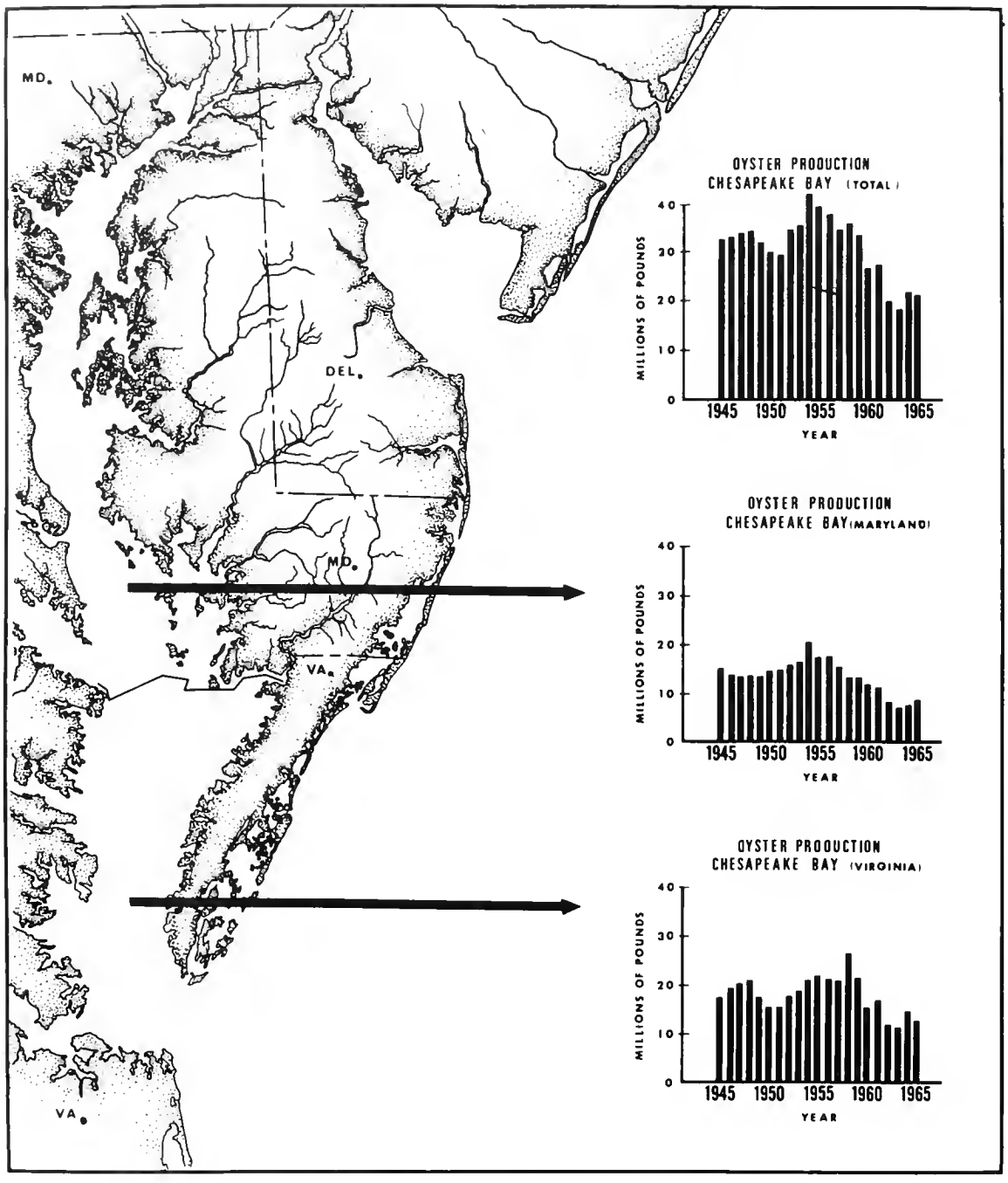

Figure 3.--Oyster production in Maryland and Virginia waters of Chesapeake Bay 1945-65.

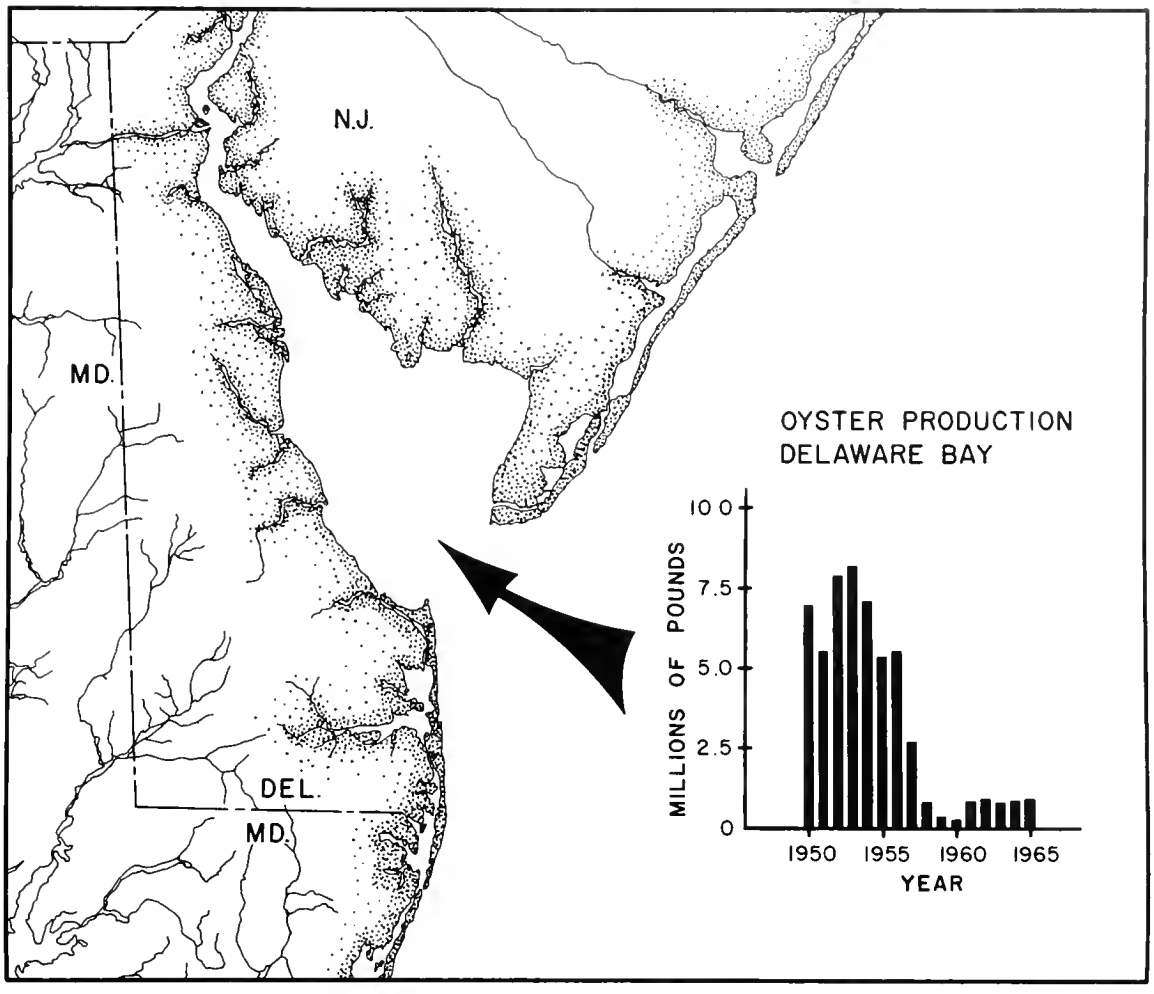

Figure 4.--Oyster production in New Jeresy waters of Delaware Bay, 1950-65.

The organism that has caused these major economic losses to the oyster industry of lower Chesapeake Bay and Delaware Bay is a haplosporidan protozoan parasite, recently named Minchinia nelsoni by Haskin, Stauber, and Mackin (1966). It was formerly known by the nickname "MSX," and the disease it causes is known as "delaware bay disease." Its most common stage in the oyster, the vegetative or plasmodial stage, is shown in figure 5. The spore stage of the pathogen (probably the stage in which the disease is transferred) was recently identified by Couch, Farley, and Rosenfield (1966). Descriptions of the disease in oyster populations have been published by Mackin (1960), Andrews (1964, 1966), and Haskin, Canzonier, and Myhre (1965).

An interesting and possibly critical aspect of the disease is that it has not been found where salinity is consistently less than 15 parts per thousand. This fact alone probably explains why the decline in Chesapeake Bay oyster production has not been as drastic as that in Delaware Bay, where salinities in oyster growing areas are higher. Virginia production

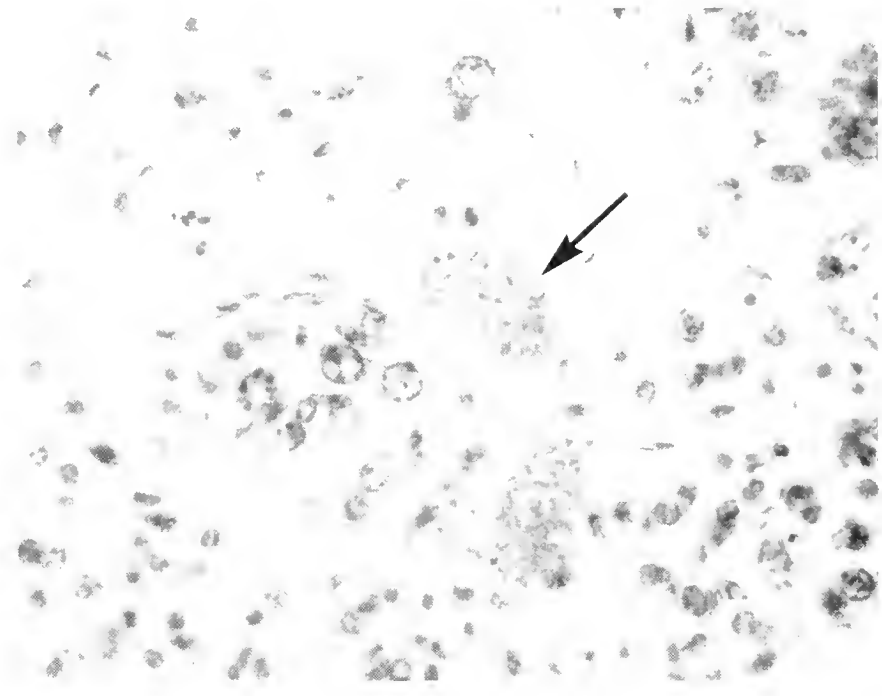

Figure 5.--Plasmodium of Minchinia nelsoni in oyster tissues, X 1,000.

has been sustained by beds in low salinities, and much of the Maryland section of the Bay, because of lower salinities, has been free of the disease--until recently. Extreme drought conditions during 1963 to 1966 pushed the "salinity barrier" of 15 parts per thousand further up Chesapeake Bay into Maryland waters (Rosenfield and Sindermann, 1966). Oyster stocks previously unaffected acquired the disease (fig 6) and suffered mortalities--thus acting to offset beneficial effects of the State's shell planting and seed transfer program.

Another critical aspect of the disease is the nature of the oyster's resistance to infection. It seems from studies carried on at the Bureau of Commercial Fisheries Biological Laboratory, Oxford, Md., and elsewhere, that survivors of the epizootic are more resistant to the disease than are unexposed populations. Possibly offspring of survivors will be more resistant than those of unexposed populations as selection pressure favoring resistance continues over a number of generations. Intensive work by the States of Maryland, Virginia, Delaware, and New Jersey, financed partly by Federal funds, is designed to develop resistant stocks of oysters and by this method to bring beds back into production sooner than might occur naturally.

Here then is a major producing segment of the American fishing industry that is seriously crippled by the single factor of disease, which is still present in epizootic form in Delaware Bay and the lower half of Chesapeake Bay. Oyster stocks have not recovered and, if left to nature, may take a number of years to do so unless steps can be taken to hasten propagation of resistant populations.

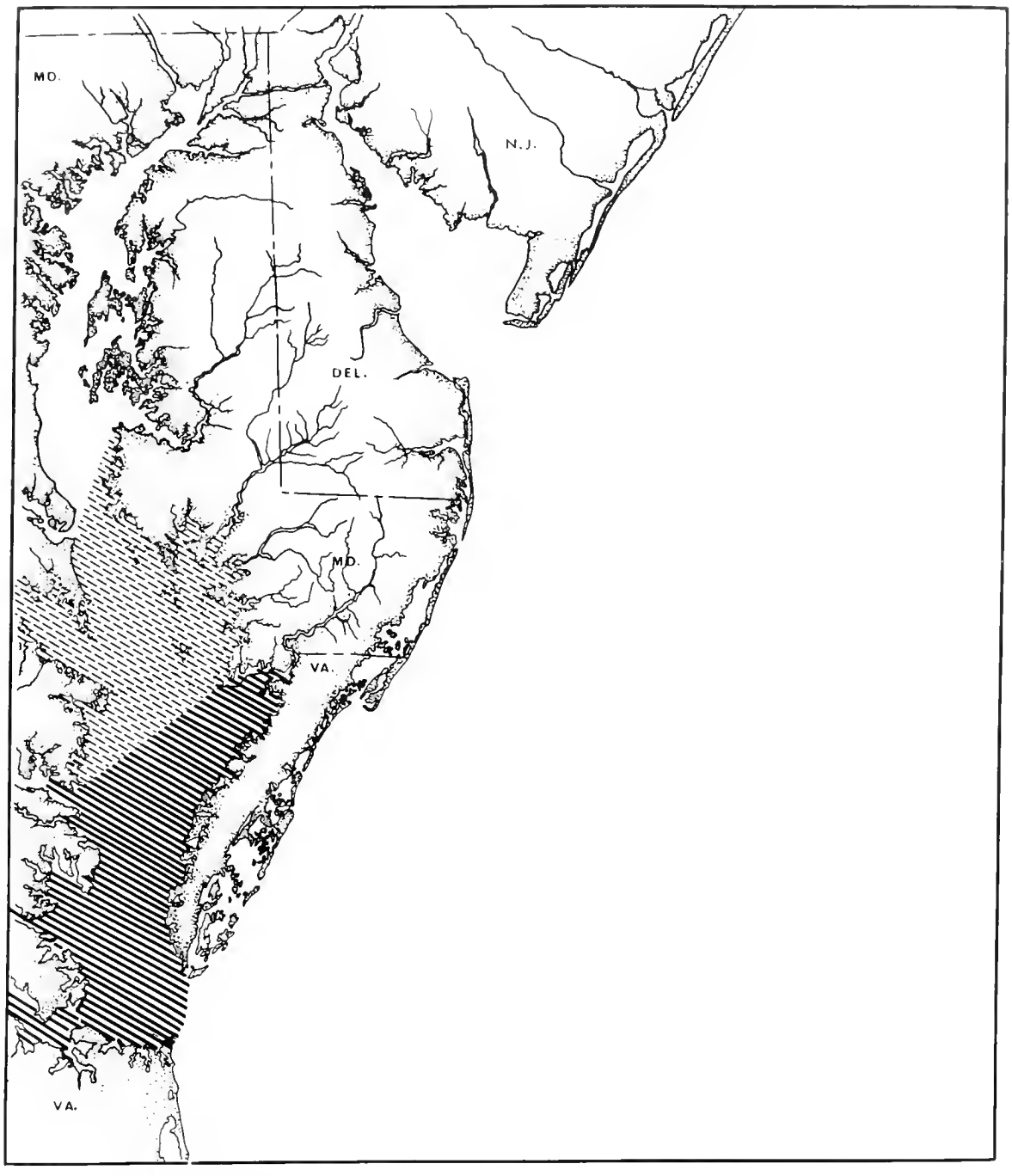

Figure 6.--Distribution of Minchinia nelsoni in Chesapeake Bay oysters in 1961 (solid hatching) and in 1966 (broken hatching).

OYSTER MORTALITIES ELSEWHERE IN NORTH AMERICA

Extensive as these recent mortalities in waters of the Middle Atlantic States have been, they form only part of a larger problem. Actually, mass mortalities of oysters have been occurring on most of the coasts of North America, and elsewhere in the world (Sindermann, 1966). American or eastern oysters are distributed from Prince Edward Island, Canada, southward to the Gulf of Mexico, with major gaps in New England. With few exceptions (such as upper Chesapeake Bay), most of these oyster populations harbor one or more serious pathogens, and many areas have had mortalities.

One disease with a history of long and frustrating scientific study was first observed in 1915 in oysters of Prince Edward Island, in the Gulf of Saint Lawrence, and has since been called "malpeque disease." From 1915 to 1933 the disease spread around the Island, destroying most of the oyster stocks--some of which required 20 years to return to previous levels of abundance (Needler and Logie, 1947; Logie, 1956). During the outbreak period, oysters apparently developed resistance to an unknown causative organism. Beginning in 1955, mortalities, probably due to the same disease, began in waters of the adjacent mainland of New Brunswick across Northumberland Strait (fig 7A). Oyster populations along the entire northern coast of New Brunswick and Nova Scotia were decimated, but mass transfer of disease-resistant oysters from Prince Edward Island waters beginning in 1957 has hastened the recovery of the fishery (Logie, Drinnan, and Henderson, 1960; Drinnan and England, 1965).

Figure 7.--The distribution of oyster diseases and pathogens on the Atlantic coast of North America: (A) malpeque disease; (B) Minchinia costalis; (C) M. nelsoni; and (D) Dermocystidium marinum.

Previously unexplained and recurring mortalities of oysters in the Gulf of Mexico were examined during the late 1940's and were found by Mackin, Owen, and Collier (1950) to be caused by a fungus, Dermocystidium marinum. Exerting its effects in higher salinities and temperatures among dense aggregations of oysters, the pathogen has been reported to cause annual mortalities in excess of 50 percent (Ray, 1954; Ray and Chandler, 1955; Mackin, 1962). It has been identified in oysters throughout the Gulf of Mexico and northward along the Atlantic Coast as far as Connecticut (fig. 7D). Although prevalences of the fungus may at times reach epizootic proportions in particular areas, its most significant effect is probably that of continuing attrition year after year during periods of high sea-water temperatures. Effects of the disease on commercial beds are now controlled to some extent by planting and harvesting at prescribed times of the year and by planting oysters thinly on beds.

In the Central Atlantic States oyster production has been affected by "delaware bay disease" caused by Minchinia nelsoni discussed earlier (fig. 7C). Even within this area, a second disease, called "seaside disease," caused by a closely related pathogen, M. costalis, has been found to kill oysters in seaside bays of Maryland and Virginia (fig. 7B) and seems to be increasing in intensity.

These mortality areas encompass a major part of the oyster producing waters of the Atlantic coast of North America--few areas are exempt. In addition, other mortalities--some of them probably disease-related--have occurred during the past decade in oyster populations on the Pacific coast of the United States and in Japan, France, and Australia.

CONCLUSIONS AND RECOMMENDATIONS

Obvious questions are: "Are these unusual occurrences? Is the 20th century a time of severe stress for oyster populations because of these great epizootics? Could this period be equated with the great human epidemics of the Middle Ages, which reduced the abundance of the human species significantly?" Although it is true that disease is always with us, and always with the oyster, and that mortalities have undoubtedly occurred in the past, new factors have been introduced by man to set the stage for the spread of epizootic disease. Oysters are transferred promiscuously from one geographic area to another; populations are often crowded in dense beds, sometimes in areas where natural populations did not exist previously; and drastic physical and chemical changes have been made in oyster habitats. We may have helped to spread diseases, and now we must control them before we can move on to higher levels of oyster culture.

Another obvious question is "What can be done when we are faced with a mortality problem as great as the one that now exists in the Middle Atlantic States?" The methods of choice include:

(1) Prevention of planting of susceptible oysters in epizootic areas and a quarantine to prevent transfer of infected individuals from such areas;
(2) development of disease-resistant strains of oysters by using hatchery techniques and also by concentrating survivors of epizootics on natural beds to serve as spawning stocks for production of resistant offspring;
(3) use of the salinity barrier that was mentioned earlier--the intolerance of the disease agent to low salinities--to prevent or possibly to eliminate infections;
(4) protection of resistant populations from fishing until adequate stocks are rebuilt;
(5) use of artificial environments, such as ponds, where disease control measures can be effected; and
(6) rapid development of basic information about the life history and ecology of the pathogen.

Some progress in the development of knowledge about diseases of oysters--and in the application of this knowledge to industry problems--has been made. Research and management agencies--Federal, State, and university--have been and are studying oyster diseases and mortalities, so progress should be rapid during the next several years.

As a final note of perspective: It is of course true that many different things--physical, chemical, and biological--can kill oysters. Any single factor, however, may become overriding at any particular time in the life of a species; here we have a clear instance in oysters of how disease can reduce the abundance of a marine species. It is likely that mass mortalities are and have always been a natural method of population control, but until recently they would have been accepted with the same dazed bewilderment and inaction that characterized the behavior of our ancestors during the plagues of the Dark Ages. We can now look to methods of environmental control and stock manipulation, particularly for sedentary shallow-water species such as the oyster, as part of the methodology in an increasingly complex system of cultivation for coastal waters.

LITERATURE CITED

ANDREWS, JAY D.
1964. Oyster mortality studies in Virginia. IV. MSX in James River public seed beds. Proc. Nat. Shellfish. Ass. 53: 65-84.

1966. Oyster mortality studies in Virginia. V. Epizootiology of MSX, a protistan pathogen of oysters. Ecology 47: 19-31.

COUCH, JOHN A., C. AUSTIN FARLEY, and AARON ROSENFIELD.
1966. Sporulation of Minchinia nelsoni (Haplosporida, Haplosporidiidae) in Crassostrea virginica (Gmelin). Science 153: 1529-1531.

DRINNAN, ROY E., and L. A. ENGLAND.
1965. Further progress in rehabilitating oyster stocks. Fish. Res. Bd. Can., Gen. Ser. Circ. Biol. Sta., St. Andrews, N. B. 48, 4 pp.

ENGLE, JAMES B.
1966. The molluscan shellfish industry; current status and trends. Proc. Nat. Shellfish. Ass. 56: 13-21.

GALTSOFF, PAUL S.
1956. Ecological changes affecting the productivity of oyster grounds. Trans. 21st N. Amer. Wildl. Conf.: 408-419.

HASKIN, HAROLD H., WALTER J. CANZONIER, and JOHN L. MYHRE.
1965. The history of "MSX" on Delaware Bay oyster grounds. Amer. Malacol. Un., Bull. 32: 20-21. (Abstract).

HASKIN, HAROLD H., LESLIE A. STAUBER, and JOHN G. MACKIN.
1966. Minchinia nelsoni n. sp. (Haplosporida, Haplosporidiidae): causative agent of the Delaware Bay oyster epizootic. Science 153: 1414-1416.

LOGIE, R. REED.
1956. Oyster mortalities, old and new, in the Maritimes. Fish. Res. Bd. Can., Progr. Rep. Atl. Coast Sta. 65: 3-11.

LOGIE, R. REED, ROY E. DRINNAN, and E. B. HENDERSON.
1960. Rehabilitation of disease-depleted oyster populations in eastern Canada. Proc. Gulf Caribbean Fish. Inst. 13th Annu. Sess: 109-113.

MACKIN, JOHN G.
1960. Status of researches on oyster diseases in North America. Proc. Gulf. Caribbean Fish. Inst. 13th Annu. Sess: 98-109.

1962. Oyster disease caused by Dermocystidium marinum and other microorganisms in Louisiana. Publ. Inst. Mar. Sci., Univ. Tex. 7: 132-229.

MACKIN, JOHN G., H. MALCOLM OWEN, and ALBERT COLLIER.
1950. Preliminary note on the occurrence of a new protistan parasite, Dermocystidium marinum n. sp. in Crassostrea virginica (Gmelin). Science 111: 328-329.

NEEDLER, A. W. H., and R. REED LOGIE.
1947. Serious mortalities in Prince Edward Island oysters caused by a contagious disease. Trans. Roy. Soc. Can. Ser. 3, Sec. V, 41: 73-89.

RAY, SAMMY M.
1954. Biological studies of Dermocystidium marinum, a fungus parasite of oysters. Rice Inst. Pam., Spec. Issue, Nov. 1954, Monograph in Biology, 114 pp.

RAY, SAMMY M., and ASA C. CHANDLER.
1955. Dermocystidium marinum, a parasite of oysters. Exp. Parasitol. 4: 172-200.

ROSENFIELD, AARON, and CARL J. SINDERMANN.
1966. The distribution of "MSX" in middle Chesapeake Bay. Proc. Nat. Shellfish. Ass. 56: 6. (Abstract).

SINDERMANN, CARL J.
1966. Epizootics in oyster populations. Proc. 11th Pac. Sci. Congr. (Fish. Sci.) 7: 7

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